AMENDMENTS TO THE CLAIMS

1. **(Original)** A method for the treatment of overproduction of mucin in a mammal, comprising: administering an inhibitor of p38 MAP kinase to the mammal in an amount sufficient to reduce mucin production.

2. **(Original)** The method of Claim 1 wherein the overproduction of mucin is caused by an otitis media (OM) infection or chronic obstructive pulmonary disease (COPD).

3. **(Original)** The method of Claim 2 wherein the OM or COPD is caused by nontypeable *Haemophilus influenzae* (NTHi).

4. **(Original)** The method of Claim 1 wherein said inhibitor of p38 MAP kinase is a chemical inhibitor selected from the group consisting of: pyridimylimidazol SB203580, SB202190, SB220025, SC68376, SKF-86002, a dominant-negative mutant of p38α, and a dominant-negative mutant of p38β.

5. **(Original)** The method of Claim 1 wherein the inhibitor of p38 MAP kinase is an antisense oligonucleotide.

6. **(Original)** The method of Claim 1 wherein the inhibitor of p38 MAP kinase is a vector which expresses a protein or polypeptide which inhibits p38 MAP kinase.

7. **(Original)** The method of Claim 1 wherein the method of administration is selected from the group consisting of: inhalation, ear drops, transtympanically, intramuscularly, intravenously, and by mouth.

8. **(Original)** A method for the identification of regulators of mucin production, comprising:

providing a reporter vector containing the MUC5AC or p38 MAP kinase promoter;

contacting the reporter vector with a potential regulator; and

identifying the up-or down-regulation of the reporter gene.

9. **(Original)** The method of Claim 8, wherein said potential regulator is selected from the group consisting of: a polypeptide, a polynucleotide, and a small molecule.

10. **(Original)** The method of Claim 8, wherein said potential regulator is a mixture of proteins from a cell.

11. **(Original)** The method of Claim 8, wherein said potential regulator is an antisense polynucleotide.

12. **(Original)** The method of Claim 8, wherein said potential regulator is a library of small molecules.

13. **(Original)** A method for the treatment of overproduction of mucin in a mammal, comprising: administering an activator of PI-3 kinase to the mammal in an amount sufficient to reduce mucin production.

14. **(Original)** The method of Claim 13 wherein the overproduction of mucin is caused by a disease selected from the group consisting of: Otitis media, chronic obstructive pulmonary disease, asthma, and cystic fibrosis.

15. **(Original)** The method of Claim 14, wherein said overproduction of mucin is caused by otitis media (OM) infection or chronic obstructive pulmonary disease (COPD).

16. **(Original)** The method of Claim 14 wherein the OM or COPD is cause by nontypeable Haemophilus influenzae (NTHi).

17. **(Original)** The method of Claim 13 wherein said activator of PI-3 kinase is a protein selected from the group consisting of: a dominant negative mutant of PI-3 kinase, a constitutively active form of p110 (p110-CAAX), wildtype Akt.

18. **(Original)** The method of Claim 13 wherein the inhibitor of p38 MAP kinase is an antisense oligonucleotide.

19. **(Original)** The method of Claim 13 wherein the inhibitor of PI-3 kinase is a vector which expresses a protein or polypeptide which activates PI-3 kinase.

20. **(Original)** The method of Claim 13 wherein the method of administration is selected from the group consisting of: inhalation, ear drops transtympanically, intramuscularly, intravenously, and by mouth.

Please add new claims:

21. **(New)** The method of Claim 1 wherein the overproduction of mucin is caused by a chronic sinusitis infection.

22. **(New)** The method of Claim 21 wherein the chronic sinusitis infection is caused by nontypeable *Haemophilus influenzae* (NTHi).